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Abbreviations: BLL (Blood Lead Level), DALY (Disability Adjusted Life Year), DW (Disability Weight), IEUBK (Integrated Exposure Uptake Biokinetic), IRIS (Integrated Risk Information System), ISS (Initial Site Screening), LMIC (Low and Middle Income Country), TSIP (Toxic Sites Identification Program), US EPA (United States Environmental Protection Agency), WHO (World Health Organization), YLD (Years Lived with Disability), YLL (Years of Life Lost)

ABSTRACT

Background: Prior calculations of the burden of disease from toxic exposures have not included estimates of the burden from toxic waste sites due to the absence of exposure data.

Objective: To develop a Disability Adjusted Life Year (DALY)-based estimate of the disease burden attributable to toxic waste sites. We focused on three low and middle income countries (LMICs) **-** India, Indonesia, and the Philippines.

Methods: Sites were identified through Blacksmith Institute's Toxic Sites Identification Program, a global effort to identify waste sites in LMICs. At least one of eight toxic chemicals were sampled in environmental media at each site and the population at risk estimated. By combining estimates of disease incidence from these exposures with population data, we calculated the DALYs attributable to exposures at each site.

Results: We estimated that in 2010, 8,629,750 individuals were at risk of exposure to industrial pollutants at 373 toxic waste sites in the three countries, and that these exposures resulted in 828,722 DALYs, with a range of 814,934 to 1,557,121 DALYs depending on the weighting factor used. This disease burden is comparable to estimated burdens for outdoor air pollution (1,448,612 DALYs) and malaria (725,000 DALYs) in these countries. Lead and hexavalent chromium collectively accounted for 99.2% of the total DALYs for the chemicals evaluated.

Conclusions: Toxic waste sites are responsible for a significant burden of disease in LMICs. While some factors, such as unidentified and unscreened sites, may cause our estimate to be an underestimate of the actual burden of disease, other factors, such as extrapolation of environmental sampling to the entire exposed population, may overestimate the burden of disease attributable to these sites. Toxic waste sites are a major, and heretofore under-recognized, global health problem.

Introduction

Toxic waste sites threaten the environment and human health in countries around the world. In developing countries these sites, and their risks to human health, have not been optimally assessed (Yáñez et al. 2002). Quantification of the burden of disease from toxic waste sites can assist public health planning and remediation efforts by complementing traditional waste site investigations and by framing these toxic exposures in the context of other exposures. Burden of disease estimates are typically expressed in Disability Adjusted Life Years (DALYs). The DALY metric accounts for both the morbidity and mortality that result from a disease, injury, or health state (Prüss-Üstün et al. 2003).

Prior calculations of the burden of disease from toxic exposures have not included estimates from toxic waste sites due to the absence of data on exposures and health impacts. In 2004, Fewtrell and colleagues estimated that lead causes nearly 1% of the global burden of disease (Fewtrell et al. 2004). Then in 2011, Prüss-Üstün et al. calculated that exposure to a variety of chemicals, including lead, second hand smoke, and asbestos, accounts for 5.7% of total global DALYs and 8.3% of total global deaths (Prüss-Üstün et al. 2011). However, because of insufficient data, neither of these studies included estimates for disease and death attributable to exposures from toxic waste sites.

We aim to develop a DALY-based estimate of the burden of disease and death attributable to toxic waste sites in India, Indonesia, and the Philippines. To our knowledge, no systematic evaluation of toxic waste sites in low and middle income countries (LMICs) has previously been performed. The resulting paucity of data has precluded calculation of the burden of disease

resulting from exposures at these sites. Through this effort we hope to ultimately calculate the contribution of toxic waste sites to the global burden of disease.

Methods

Site Identification

This paper utilizes data collected through Blacksmith Institute's Toxic Sites Identification Program (TSIP), an effort to identify and screen contaminated sites in LMICs (Blacksmith Institute 2013). The TSIP, which is implemented jointly with the United Nations Industrial Development Organization, identifies point-source pollution from industrial sites that present a public health risk. A particular focus is placed on abandoned, "legacy", sites, such as former tanneries, as well as small-scale artisanal sources, such as lead battery recycling and artisanal gold mining. While other sources of contamination, such as large scale mining, may also be included and screened, the majority of sites come from these two categories (i.e., legacy sites and artisanal sources). The TSIP excludes non-point sources, such as ambient urban air pollution, and non-chemical contamination, such as sewage-contaminated water. Ericson et al. (2012) described the types of sites identified in the TSIP.

Blacksmith Institute developed an evaluation instrument, the Initial Site Screening (ISS), for rapid data collection and assessment of these sites (Blacksmith Institute 2013). The ISS is a modified and simplified version of the US EPA's Hazard Ranking System, used to prioritize and rank toxic waste sites in the US EPA's Superfund program (EPA 2012b). The ISS includes information on the concentration of the key toxic chemical, the primary environmental medium of the exposure pathway, and the size of the population at risk.

To undertake the TSIP, Blacksmith Institute contracted and trained approximately 150 site investigators. These investigators identified and visited sites, collected environmental samples, took photographs and GPS coordinates, interviewed stakeholders, and categorized the potential contaminated environmental media. After being educated on the project and ensured that participation in the interview process was voluntary, the stakeholders agreed to participate; written informed consent was not obtained. The investigators determined the dominant pollutant for each site based in part on prior testing or historical use of a site, then took samples to measure levels of the pollutant, typically in only one environmental medium. For sites where only total chromium was reported, the speciation coefficient of 0.6 was used to estimate hexavalent chromium (Avudainayagam et al. 2003; Kumar and Riyazuddin 2010). Between 2009 and 2012, investigators completed 1,510 such screenings in 49 countries. Since the majority of screenings occurred in 2010, we used 2010 as our baseline year for analysis. We have previously described the ISS protocol and TSIP in detail (Ericson et al. 2012).

Population at Risk of Exposure

As part of the ISS, investigators estimated the population at risk of exposure for each site, indicating the number of people regularly coming into contact with the contaminant in the relevant environmental medium. For example, if water contamination is documented, then the population at risk includes those individuals who utilize the water daily for drinking, food preparation, and other domestic uses. Investigators used a range of approaches to obtain this information, including visual methods, satellite photographs, community census data, government interviews, and personal knowledge. The age distribution at sites was not recorded as part of the ISS. Therefore, we applied age distribution estimates from the US Census Bureau (2012) for each country to the population around each site within that country. We divided each

site's estimated population into 17 age groups based on these distributions (0-4, 5-9, 10-14, etc.). The World Health Organization (WHO) DALY calculator for cardiovascular disease resulting from adult lead exposure uses five age groups (15-29, 30-44, 45-59, 60-69, 70-79). In this instance, the 17 age groups were condensed into the appropriate five groups to enable these calculations.

Calculating Risk Per Person

We divided human health effects into cancer and non-cancer effects. For carcinogens, we used the US EPA's Regional Screening Level Calculator for Chemical Contaminants to calculate long-term cancer risk per unit toxicant (i.e., cancer probability per milligram/kilogram for agents found in soil or microgram/liter for waterborne agents) (EPA 2012c). For non-cancer health effects, reference doses (RfD) and concentrations (RfC) from the US EPA's Integrated Risk Information System (IRIS) database were applied to the exposure pathways and contamination levels at each site (EPA 2012a). The modeling assumed a linear dose-response and used the health outcome associated with the RfD or RfC (e.g., liver toxicity, renal toxicity). A listing of the cancer and non-cancer risks per unit of contaminant, with the exception of lead, is presented in Table 1. Given the availability of lead-specific modeling tools and dose-response relationships, we calculated disease incidence and DALYs from lead separately.

Calculating Incidence of Disease

For each chemical we considered up to three environmental media (soil, water, air) and corresponding routes of exposure (ingestion, dermal, and/or inhalation). To calculate disease incidence for all chemicals with the exception of lead, we multiplied the risk per person by the level of the contaminant in the relevant environmental medium. Because linear slope factors were utilized to calculate incidence, very high concentrations of contaminants resulted in

correspondingly high estimates of disease incidence. To accommodate this limitation of the model, we arbitrarily capped incidence for all diseases at five percent.

For lead, we calculated the incidence of mild mental retardation and anemia in children, and cardiovascular disease in adults resulting from lead-induced increases in blood pressure. We calculated the predicted mean blood lead levels (BLLs) that would result from lead exposures at each site by entering the soil and drinking water lead levels measured at each site into the US EPA's Integrated Exposure, Uptake and Biokinetic Model for Lead (IEUBK) and Adult Lead Methodology (ALM) (Caravanos et al. 2012; EPA 1994; White et al. 1998). We calibrated default soil ingestion levels in the IEUBK model upward from 200 mg/day to 400 mg/day. This approach follows similar analyses done in Native American populations (400 mg/day) as well as indigenous populations in Micronesia (500 mg/day), and is above the "upper bound" level (200 mg/day) utilized by the US EPA (EPA 2011; Harris and Harper 2004; Sun and Meinhold 1997). Then we calculated the incidence of mild mental retardation and cardiovascular outcomes that would result from such BLLs, utilizing spreadsheets developed by the WHO (WHO 2013). We also assumed that 20% of children with BLLs greater than 70 μg/dL develop anemia (Fewtrell et al. 2003).

Calculating YLD and YLL

The DALY metric is the sum of two components – Years Lived with Disability (YLD), which represent disease-related morbidity, and Years of Life Lost (YLL), which represent the premature mortality from the disease. We calculated YLD and YLL for exposure to each contaminant through each relevant environmental medium. YLD is the product of the estimated years lived with a given disability multiplied by its specific Disability Weight (DW). The DW is a value from zero to one depending on the severity of each disease, with zero representing ideal

health and one representing death. For example, periodontal disease has a DW of 0.001, while a first-time stroke has a DW of 0.920 (WHO 2008).

For each chemical we assigned the relevant type of cancer, non-cancer health effect, and corresponding DW (Table 2) (EPA 2012a; WHO 2008). If the chemical's health effect did not align with a disease in the WHO DW database, then we selected the most appropriate disease and DW based on the target organ, duration of disease, and severity of disease. In the case of non-carcinogenic effects, the total number of years of life remaining at onset was multiplied by the appropriate DW to determine YLD (Prüss-Üstün et al. 2003). We chose to apply the exposure for the remainder of an individual's life expectancy given that most LMICs do not have a systemic program to identify and remediate these sites. For carcinogens we applied a DW and duration to each cancer stage – Diagnosis (cancer-specific DW; three years); Metastasis (DW 0.75; one year); and Terminal (DW 0.81; one year) (WHO 2008). YLL, which indicate the number of years of life lost due to premature death from disease, were calculated only for carcinogens. We used cancer incidence and survival data to calculate the resulting number of deaths (Ferlayet al. 2010; Sankaranarayanan et al. 2010). All cancers were assumed to last five years, before either going into remission or resulting in death.

For lead, we utilized the environmentally attributable fraction approach in determining the contribution of lead exposure to the burden of cardiovascular disease (ischemic heart disease, cerebrovascular disease, hypertensive disease, and other cardiac disease) (Fewtrell et al. 2003). The WHO has calculated the fraction of cardiovascular disease attributable to lead exposure based on the BLL. By entering the predicted BLL and total cardiovascular disease DALYs for each country into a WHO spreadsheet, we calculated the DALYs attributable to cardiovascular disease from lead exposure at toxic waste sites in each of the three countries. In addition, for

children with BLLs greater than 10 µg/dL who did not have mental retardation, we applied the DW for developmental disability from protein-energy malnutrition (0.024) as a proxy DW for lifelong disability from IQ loss in the absence of mental retardation. Prior research suggests that the loss of IQ points may impact cardiovascular and all-cause mortality, resulting in increased morbidity and mortality (Batty et al. 2010; Lager et al. 2009).

We then applied weighting factors to the resulting YLL and YLD for each chemical, including a discount rate to account for inherent inaccuracies when predicting future events, and age weights to reflect the relative societal value of different age groups (Mathers et al. 2006). The notation $DALYs_{(r,K)}$ signifies which discount rate (r) and age weight (K) is used. Our primary results are expressed as $DALYs_{(3,1)}$, which include a 3% discount rate and the full age weight. We also calculated $DALYs_{(3,0)}$ with the 3% discount rate only, and $DALYs_{(0,0)}$ without any weighting to provide a range of estimates (Mathers et al. 2006).

For example, the drinking water at one site in India had an Aldrin level of 0.063 ppb. The oral RfD for Aldrin for liver toxicity is 3.0×10^{-5} mg/kg/day, which converts to a risk of 8.57×10^{-10} per µg/L of drinking water (EPA 2012a; EPA 2012c). The DW for advanced hepatic disease is 0.104. Assuming the 4,000 individuals potentially exposed consume two liters of drinking water each day, we calculated 1.62 $DALYs_{(3,0)}$, 1.64 $DALYs_{(3,1)}$, and 2.02 $DALYs_{(0,0)}$ resulting from exposure to Aldrin in drinking water at this site.

Sensitivity Analysis

In addition to calculating DALYs with varying rates and weights, we also altered inputs into our model to conduct a sensitivity analysis. We varied the total population at risk by 25%, changed the disease incidence cap from the default value of 5% to 2.5% or 7.5%, and removed the

additional DW for lead-induced IQ losses that did not result in mild mental retardation. For a remediation scenario, we also assumed that remediation had reduced all pollutants to concentrations below international standards (Blacksmith Institute 2011). By subtracting the resulting DALYs from our primary estimate, we quantify the potential impact of remediating these sites.

We also estimate that an additional 5,000 unscreened sites exist in these countries, and that these sites present similar conditions as the screened sites. The TSIP prioritized screenings in part by the scale of the problem, measured in population at risk. Thus, these 5,000 sites are unlikely to have comparably large populations. We therefore assumed that the population at risk for each of these additional sites was the median of the population at risk of screened sites, which is lower than the mean population for screened sites. By contrast, the DALY per person estimates for the 5,000 unscreened sites are unlikely to be lower than those identified at the screened sites. Sites were not prioritized for screening based on the level of the contaminant in the pathway. Therefore, we applied the average DALY per person for the screened sites to the population at the unscreened sites.

Results

Sites Evaluated

Blacksmith Institute-trained investigators screened 498 sites in India, Indonesia, and the Philippines with an estimated population at risk of exposure of approximately 12 million. Of the 23 separate chemicals documented at these sites, eight occurred at more than one site and had established dose-response relationships correlating exposure with specific outcomes. We included in the analysis only the 373 sites with one of these eight chemicals. Figure 1 displays

the geographical distribution of the sites in India (n = 221), Indonesia (n = 73), and the Philippines (n =79). The estimated population at risk of exposure at these 373 sites was 8,629,750 (mean = 23,136, median = 7,000), which is 0.61% of the total population of the three countries. Of the exposed population, 3,449,592 are younger than 18 years of age and 2,184,220 are women of childbearing age (15-49 years of age). We estimate that an additional 5,000 unscreened sites exist in the three countries, with a population of 7,000 individuals per site. This additional population equals 35,000,000, resulting in a total population of 43,629,750 for the screened and unscreened sites.

YLD and YLL at Screened Sites

We estimated 588,112 person-years lived with disease and 240,610 person-years lost as a result of chemical exposures in 2010 at the 373 toxic waste sites (Table 3). According to our estimates, lead was the largest contributor of the 8 chemicals to YLD (523,630 YLD, 89% of total YLD), and hexavalent chromium was the largest contributor to YLL (235,483 YLL, 97.9% of total YLL). In Table 3, inhalation of soil and dust is incorporated into the soil results.

Premature Deaths and DALYs at Screened and Unscreened Sites

We estimated that 828,722 $DALYs_{(3,1)}$ resulted from chemical exposures at the 373 sites in 2010. By applying the value of 0.10 $DALYs_{(3,1)}$ per person from the screened sites to the population at the unscreened sites, we estimated that 3,500,000 $DALYs_{(3,1)}$ resulted from exposure at the unscreened sites. The total estimated $DALYs_{(3,1)}$ for the screened and unscreened sites was 4,328,722. We also calculated that 66,747 individuals would die prematurely from cancer, specifically liver and lung cancer, from exposures at these sites.

Sensitivity Analysis

Removal of age weights yielded 814,934 $DALYs_{(3,0)}$, while removal of age weights and the discount rate yielded 1,557,121 $DALYs_{(0,0)}$ (Table 4). If the actual exposed population around these sites is 25% less or 25% more than our estimate, the resulting $DALYs_{(3,1)}$ would be 621,541 and 1,035,902, respectively. If the additional DW for lead-induced IQ loss not resulting in mental retardation is removed, our overall estimate would be 483,201 $DALYs_{(3,1)}$. In addition, if disease incidence is capped at 2.5% or 7.5%, the resulting $DALYs_{(3,1)}$ would be 730,627 and 922,479, respectively. The remediation scenario yielded 30,317 $DALYs_{(3,1)}$, in contrast with our primary estimate of 828,722 $DALYs_{(3,1)}$. Thus, our estimates suggest that 798,405 $DALYs_{(3,1)}$ could be eliminated by remediation of these sites to achieve international standards.

Discussion

We estimated that 8,629,750 individuals were at risk of exposure to one of eight industrial pollutants at 373 toxic waste sites in three countries in 2010, resulting in 828,722 $DALYs_{(3,1)}$. This estimate represents a burden of disease equal to 0.22% of the total estimated $DALYs_{(3,1)}$ from all causes in India, Indonesia, and the Philippines (WHO 2008). Alteration of the discount rate and age weight leads to a range of estimates, from 814,934 $DALYs_{(3,0)}$ to 828,722 $DALYs_{(3,1)}$ to 1,557,121 $DALYs_{(0,0)}$. Lead and hexavalent chromium account for 99.2% of the total DALYs estimated for the 8 waste site chemical exposures evaluated. The additional DW for lead-induced IQ loss not resulting in mental retardation accounts for 483,201 $DALYs_{(3,1)}$, which represents approximately 58% of total $DALYs_{(3,1)}$. Inclusion of an estimated number of unscreened sites increased the estimated population at risk of exposure to 43,629,750, and the total $DALYs_{(3,1)}$ to 4,328,722. As part of a larger project attempting to calculate the burden of

disease of toxic waste sites in LMICs, the present analysis indicates that the burden of disease associated with these sites is substantial and comparable to well-described diseases and environmental risk factors. For example, the WHO (2009) estimates that outdoor air pollution causes 1,448,612 $DALYs_{(3,1)}$ and malaria causes 725,000 $DALYs_{(3,1)}$ in these three countries. Overall, the present analysis begins to address the paucity of knowledge regarding health effects from toxic waste sites in LMICs and helps frame this issue in the context of other public health problems.

Given the limited scope of this project and the understanding that the screened sites represent only a portion of the total existing sites, we estimated that 5,000 unscreened sites exist in these three countries. The US EPA (2004) estimates that there are approximately 294,000 contaminated sites in the United States alone that require some form of remediation. India's population is nearly four times that of the US, with nearly one third of Indian urban residents living in informal housing settlements, where unregulated cottage industries can proliferate without zoning or emissions controls (UN-HABITAT 2007).

Pollutants at toxic waste sites in LMICs can potentially have profound health effects. Lead and cadmium adversely affect neurodevelopment in children, with the *in-utero* period being the life stage of greatest vulnerability (Ciesielski et al. 2012; Hu et al. 2006). Children and women of childbearing age constitute 65.3% of the total exposed population in this analysis, highlighting the potential impact on these vulnerable populations. The majority of the chemicals are nephro- or hepatotoxic, and kidney and liver toxicity accounted for the majority of non-cancer health effects. Several are known carcinogens, including asbestos, cadmium, and chromium.

Prior work has described the difficulty in identifying which toxic chemicals are being generated in India via industrial processes, as well as which ones are being imported for recycling or disposal (Dutta et al. 2006). The actual amount being produced and imported, and the ultimate fate of many of these chemicals, is unclear. Misra and Pandey (2005) discuss the complex requirements for proper handling of toxic waste to prevent human exposures and highlight the barriers to achieving this goal in countries such as India. Waste is often handled without adequate control mechanisms, such as proper infrastructure and personal protective equipment, in dense, highly populated areas, exposing not only workers but also residents in the surrounding communities.

Our estimates highlight the need for remediation of these sites, with a focus on addressing the key pollutant and dominant environmental medium. High dose, mass poisonings periodically come to worldwide attention, such as recent events in Nigeria and Senegal, prompting immediate focus and remediation (Dooyema et al. 2012; Haefliger et al. 2009). However, exposures from most toxic waste sites continue unabated. Research has documented that waste site remediation can be cost-effective while reducing toxic exposures (Guerriero et al. 2011; Jones et al. 2011).

We must note several limitations of this analysis. We examined only eight chemicals and restricted the analysis to only one chemical per site. Individuals living near toxic waste sites are often exposed to multiple chemicals simultaneously (DeRosa et al. 1996; Hu et al. 2007; Vrijheid 2000). Therefore, health effects may be increased or decreased due to the existence of co-exposures and the potential for synergistic or antagonistic effects. For example, Claus Henn et al. (2011) documented a synergistic effect between lead and manganese in a Mexico City pregnancy cohort, with the impact of lead on child neurodevelopment increased in the group with higher levels of manganese.

For most of the chemicals we assigned only one cancer and one non-cancer health effect. In addition, only a limited number of diseases have an associated DW, which prevented the inclusion of some health effects. For example, exposure to hexavalent chromium can cause nasal perforation. However, there is no DW for nasal perforation, so this health effect was not included in the analysis. In several cases there were not specific DWs that aligned properly with the projected health effect. Since there is no DW for liver toxicity, for example, we applied the DW for advanced hepatic disease to those chemicals known to cause liver toxicity. While the major health effect of mercury is the impact of *in-utero* methylmercury exposure on neurodevelopment, we were unable to capture this health effect for various reasons (e.g., limited methylmercury samples, no methylmercury biomonitoring). An additional source of uncertainty is the calculation of YLD for cancer, in which each cancer stage was assigned a different duration and DW.

Limited environmental sampling occurred at most sites, forcing us to extrapolate results of several samples to the entire population at risk. Biomarkers of exposure were not obtained, so we were unable to confirm completed pathways of exposure. In the case of lead, we attempted to offset this limitation by utilizing the US EPA's IEUBK Model and ALM, which predict BLLs expected as a consequence of environmental lead exposure. However, these models may overestimate BLLs when predicted BLLs are greater than 30 μg/dL given the uncertainty in the relationship between environmental lead levels and BLLs at this level (Hogan et al. 1998). It is also likely that the actual exposures to the pollutants vary, with some individuals being exposed to lower levels. Despite evidence of prenatal exposure to environmental toxicants causing adverse health effects (Wigle et al. 2008), the current analysis did not account for effects of prenatal exposures other than lead.

In addition, we assumed that exposures continued for a lifetime since there are not established waste site remediation programs in most LMICs. While complete elimination of the toxic exposure may not be feasible for each site, a reduction in high-level exposure would decrease our disease burden estimates. Remediation of all sites such that pollutant concentrations are below international standards could save 798,405 $DALYs_{(3,1)}$. Finally, a key limitation of this analysis is its reliance on slope factors, reference doses, and reference concentrations, largely based on animal testing. These regulatory values may overestimate the disease burden given the limitations of animal testing and the assumptions required to extrapolate toxicity data from animals to humans (e.g., applying uncertainty factors). Acknowledging these limitations, we believe this paper presents the best possible estimate of the burden of disease from these sites given current data.

Further research should better define the specific exposures occurring at toxic waste sites in LMICs by linking environmental sampling levels, biomarkers of disease, and health outcomes, and focusing on uniquely vulnerable populations such as pregnant females, children, and the elderly. Such enhanced surveillance data will help provide context when comparing toxic waste sites with more recognized public health threats. This research should not preclude the immediate remediation of existing sites given the disease and resulting costs to society that result from such exposures. Given that the majority of the DALYs estimated for the 8 chemicals evaluated were due to lead and chromium exposures, remediation could be facilitated by selectively targeting lead- and chromium-contaminated sites.

Conclusions

In summary, this study documents that chemical pollutants from toxic waste sites are a large, and heretofore insufficiently, studied public health problem in the three low and middle income Asian countries that we examined - India, Indonesia, and the Philippines. Disease and death caused by toxic chemicals contribute to the total burden of disease in these countries. We estimate that more than eight million persons in these countries suffered disease, disability, or death resulting from exposures to industrial contaminants in 2010, resulting in 828,722 $DALYs_{(3,1)}$. These findings underscore the urgent need for toxic waste sites around the world to be characterized and remediated and for the health of affected populations to be monitored.

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Table 1. Per capita cancer and non-cancer human health risks by chemical and media for chemicals other than lead

Chemical and Media Assessed	Cancer Risk per ug/m^3 in Air	Cancer Risk per mg/kg in Soil	Cancer Risk per ug/l in Water	Non-Cancer Risk per ug/m^3 in Air	Non-Cancer Risk per mg/kg in Soil	Non-Cancer Risk per ug/l in Water
Aldrin (W)	N/A	N/A	5.35E-04	N/A	N/A	2.22E-06
Asbestos (A)	2.30E-01[a]	N/A	N/A	N/A	N/A	N/A
Cadmium (A, S,W)	1.80E-03	N/A	N/A	5.00E-05	2.67E-08	1.33E-07
Chromium VI (A,S,W)	8.40E-02	9.71E-8[b]	2.09E-05	N/A	N/A	N/A
DDT (W)	N/A	N/A	1.07E-05	N/A	N/A	1.33E-07
Lindane (S,W)	N/A	5.08E-06	3.45E-05	N/A	8.85E-08	2.22E-07
Mercury, inorganic (A,S,W)	N/A	N/A	N/A	5.68E-08	8.85E-08	2.22E-07

Abbreviations: A = Air, S = Soil, W = Water

[a] fibers/cubic centimeter

[b] inhaled airborne dust

Table 2. Cancer and non-cancer health effects and disability weights of chemicals found at waste sites

Chemical	Cancer Site (Classification)[a]	Cancer-specific Disability Weight[b]	Health Effect (Non-Cancer)	Disability Weight (Non-Cancer)
Aldrin (W)	Liver (probable)	0.20	Liver toxicity	0.104[c]
Asbestos (A)	Lung (confirmed)	0.15	N/A	N/A
Cadmium (A)	Lung (probable)	0.15	N/A	N/A
Cadmium (W,S)	N/A	N/A	Renal toxicity	0.091[d]
Chromium VI (A,W,S)	Lung (confirmed)	0.15	N/A	N/A
DDT (W)	Liver (probable)	0.20	Liver toxicity	0.104[c]
Lead (A, W, S)	N/A	N/A	Mild mental retardation	0.361
			Decrement in IQ	0.024[e]
			Cardiovascular disease	N/A[f]
			Anemia	0.024
Lindane (W, S)	Liver (possible)	0.20	Liver toxicity	0.104[c]
Inorganic mercury (W, S)	N/A	N/A	Renal toxicity	0.091[d]

Abbreviations: A = Air, S = Soil, W = Water, DDT = Dichlorodiphenyltrichloroethane

[a] Human carcinogenicity classification (EPA 2012a)

[b] Cancer-specific DW was applied for a duration of three years, then a DW of 0.75 was applied for one year (metastasis), followed by a DW of 0.81 for one year (terminal stage)

[c] Advanced hepatic disease

[d] Acute glomerulonephritis

[e] Developmental disability associated with protein-energy malnutrition

[f] DALYs calculated with the environmental attributable fraction approach

Table 3. Years lived with disability, years of life lost, and DALYs by chemical -

Chemical	Number of Sites	Estimated Population at Risk	Years Lived with Disability (YLD)	Years of Life Lost (YLL)	Disability Adjusted Life Years (DALYs)
Aldrin	5	133,000	212	812	1,024
Asbestos	3	25,000	974	4,218	5,192
Cadmium	53	976,600	15 (S=1, W=14)	0	15
Chromium VI	128	3,231,750	63,174 (S=3,582, W=59,592)	235,483 (S=14,467, W=221,016)	298,657
DDT	4	180,000	4	18	22
Lead	79	1,829,900	523,630	0	523,630
Lindane	9	131,300	20 (S=2,W=18)	79 (S=6,W=73)	99
Mercury, Inorganic	92	2,122,200	83 (S=32,W=51)	0	83
Total	373	8,629,750	588,112	240,610	828,722

Abbreviations: S = Soil, W = Water -

Table 4. Sensitivity analysis estimates -

Scenario	Total DALYs
Primary estimate of screened sites	828,722 $DALYs_{(3,1)}$
Estimate without age weights	814,934 $DALYs_{(3,0)}$
Estimate without age weights or discount rate	1,557,121 $DALYs_{(0,0)}$
Remediation scenario	30,317 $DALYs_{(3,1)}$
If actual exposed population is 25% less	621,541 $DALYs_{(3,1)}$
If actual exposed population is 25% greater	1,035,902 $DALYs_{(3,1)}$
If additional DW for lead-induced IQ loss not resulting in MMR is removed	483,201 $DALYs_{(3,1)}$
If incidence is capped at 2.5%	730,627 $DALYs_{(3,1)}$
If incidence is capped at 7.5%	922,479 $DALYs_{(3,1)}$
Estimate of unscreened sites	3,500,000 $DALYs_{(3,1)}$
Estimate of screened and unscreened sites	4,328,722 $DALYs_{(3,1)}$

Figure Legend

Figure 1. Locations of 373 toxic waste sites in India, Indonesia, and the Philippines in 2010. -